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Applicant Initiated Interview Request Form

Application No.: 10/561,015-Conf. #9430 First Named Applicant: Dan P. Felsenfeld
Examiner: C. Y. Wang Art Unit: 1649 Status of Application: Published

Tentative Participants:

(1) S. Peter Ludwig (2) Dan P. Felsenfeld (5) William Chiang ☐
(3) Irina E. Vainberg (4) Sybil Lombillo

Proposed Date of Interview: December 8, 2009 **Proposed Time:** 11 AM **(AM/PM)**

Type of Interview Requested:

(1) [X] **Telephonic** (2) [ ] **Personal** (3) [ ] **Video Conference**

Exhibit To Be Shown or Demonstrated: [X] **YES** [ ] **NO**

If yes, provide brief description: Attached are (i) proposed amendments to the claims and (ii) schematic diagrams (Figures 1-3) demonstrating the differences in the mechanism of action of the peptides of the invention and the full-length FIGQY->F mutant neurofascin disclosed in the Tuvia and Garver references

Issues To Be Discussed

Issues (Rej., Obj., etc)	Claims/ Fig. #s	Prior Art	Discussed	Agreed	Not Agreed
(1) 35 USC § 112	20 and 24		[X]	[ ]	[ ]
(2) 35 U.S.C. § 102(b)	6, 18, 19, 21, and 22	Tuvia or Garver	[X]	[ ]	[ ]
(3) 35 U.S.C. § 103(a)	6, 7, 18, 19, and 21-23	Tuvia or Garver	[X]	[ ]	[ ]

Brief Description of Arguments to be Presented:

To address the rejections under 35 U.S.C. § 112, first paragraph, applicants propose to amend claims 6, 7, 19, 20, 23, and 24 to replace the article "an" with "the".
With respect to the rejections under 35 U.S.C. §§ 102(b) and 103(a), applicants intend to explain to the Examiners that (i) the rejected claims do not encompass the full-length FIGQY->F mutant neurofascin of the Tuvia and Garver references and (ii) based on all available data, the full-length FIGQY->F mutant neurofascin disclosed in the Tuvia and Garver references would inhibit, not promote neurite outgrowth, if expressed as a transgene in neurons.

An interview was conducted on the above-identified application on Dec 8, 2009 .

NOTE:
This form should be completed by applicant and submitted to the examiner in advance of the interview (see MPEP §713.01).
This application will not be delayed from issue because of applicant's failure to submit a written record of this interview. Therefore, applicant is advised to file a statement of the substance of this interview (37 CFR 1.133(b)) as soon as possible.

/Chang-Yu Wang/
Applicant/Applicant's Representative Signature — Examiner/SPE Signature

Irina E. Vainberg
Typed/Printed Name of Applicant or Representative

48,008
Registration Number, if applicable

PROPOSED AMENDMENTS TO THE CLAIMS
(FOR DISCUSSION AT THE INTERVIEW 12-08-09)

1. (Withdrawn) A method for promoting outgrowth of a mammalian neuron comprising contacting said neuron with the peptide of claim 6.

2. (Withdrawn) A method for promoting extension of a mammalian neuronal cell across a substrate comprising contacting said neuronal cell with the peptide of claim 6.

3. (Withdrawn) A method for treating a diseases characterized by axonal damage, which comprises administering to a mammal in need of such treatment an effective amount for treating said diseases of the peptide of claim 6.

4. (Withdrawn) The method of claim 3, wherein the mammal is a human.

5. (Canceled)

6. (Currently Amended) An isolated peptide derived from the ankyrin binding domain of an L1-CAM family member protein, wherein said peptide comprises~~ing~~ the~~an~~ amino acid sequence of QFNEDGSFIGQF (SEQ ID NO: 2) and ~~, wherein said peptide~~ promotes neurite outgrowth.

7. (Currently Amended) The peptide of claim 6 comprising the~~an~~ amino acid sequence of QFNEDGSFIGQF (SEQ ID NO: 2) linked to the~~an~~ amino acid sequence of RQIKIWFQNRRMKWKK (SEQ ID NO: 6), wherein said sequences are linked by a disulfide bond.

8. (Withdrawn) An isolated nucleic acid encoding the peptide of claim 6.

9. (Withdrawn) A method of inhibiting neuronal signaling in a mammal which comprises disrupting the interaction between L1-CAM, ankyrin, and voltage-gated calcium channels, by contacting a neuron with thea peptide of claim 6.

10. (Withdrawn) A method for treating pain in a mammal comprising administering to the mammal an amount effective for the treatment of pain of thea peptide of claim 6.

PROPOSED AMENDMENTS TO THE CLAIMS
(FOR DISCUSSION AT THE INTERVIEW 12-08-09)

11. (Withdrawn) The method of claim 10 wherein said pain is chronic pain.

12. (Withdrawn) The method of claim 10 wherein said mammal is a human.

13. (Withdrawn) The method of claim 10, which comprises administering the peptide locally in the vicinity of the affected neurons.

14. (Withdrawn) The method of claim 10, which comprises administering the peptide with an osmotic pump.

15. (Withdrawn) The method of claim 14, which comprises situating the osmotic pump for administration of the peptide to a region of the dorsal spinal cord.

16. (Withdrawn) A method for preventing neuronal cell death after an ischemic attack or stroke in a mammal comprising administering to the mammal an amount effective for the prevention of neuronal cell death of the peptide of claim 6.

17. (Withdrawn) A method for blocking neuronal calcium flux in a mammal comprising disrupting the interaction between L1-CAM, ankyrin, and voltage gated calcium channels by contacting a neuron with the peptide of claim 6.

18. (Previously Presented) A pharmaceutical composition comprising the peptide of claim 6 and a pharmaceutically acceptable carrier.

19. (Currently Amended) An isolated peptide consisting essentially of thean amino acid sequence of QFNEDGSFIGQF (SEQ ID NO: 2), wherein said peptide promotes neurite outgrowth.

20. (Currently Amended) The peptide of claim 19 consisting of thean amino acid sequence of QFNEDGSFIGQF (SEQ ID NO: 2).

21. (Previously Presented) A pharmaceutical composition comprising the peptide of claim 19 and a pharmaceutically acceptable carrier.

PROPOSED AMENDMENTS TO THE CLAIMS (FOR DISCUSSION AT THE INTERVIEW 12-08-09)

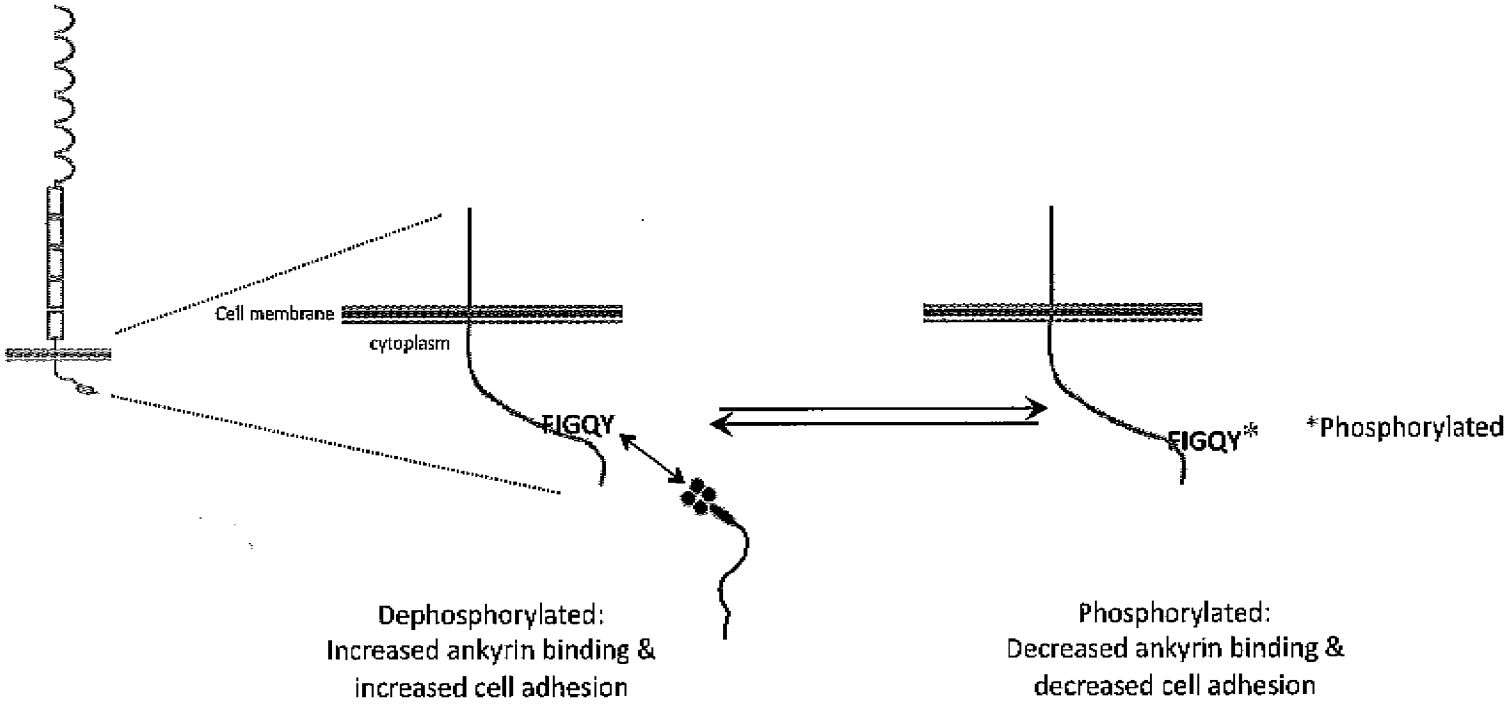
22. (Previously Presented) The peptide of claim 6 which comprises a targeting sequence which allows translocation of the peptide across the plasma membrane and into the cytoplasm of cells.

23. (Currently Amended) The peptide of claim 22, wherein the targeting sequence comprises the~~an~~ amino acid sequence of RQIKIWFQNRRMKWKK (SEQ ID NO: 6).

24. (Currently Amended) The peptide of claim 23 consisting of the~~an~~ amino acid sequence of RQIKIWFQNRRMKWKKQFNEDGSFIGQF (SEQ ID NO: 3).

25. (Withdrawn) The method of claim 3, wherein the disease is selected from the group consisting of spinal cord injury, traumatic brain injury, stroke, and neurodegenerative disease.

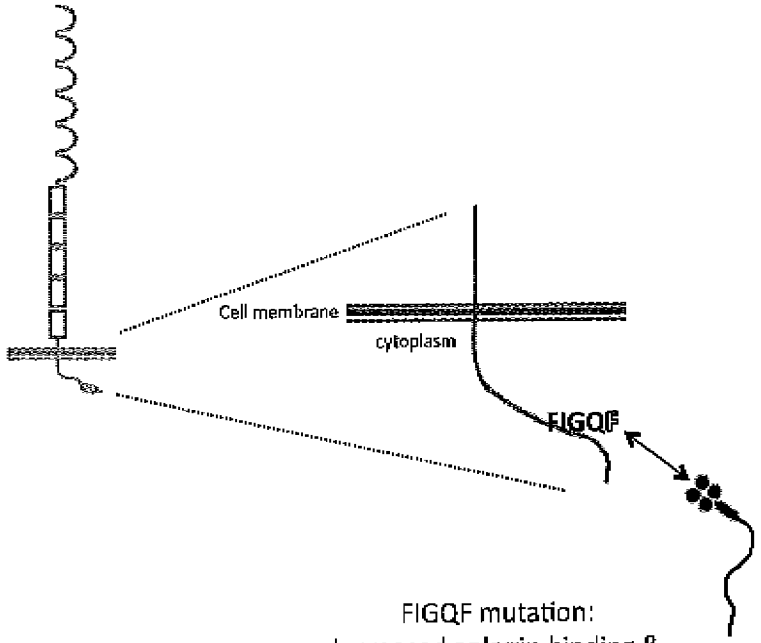
Figure 1. Schematic diagram showing transmembrane & cytoplasmic domain of full-length wild-type L1 family members



- FIGQY phosphorylation inhibits ankyrin binding to L1 family members;
- Ankyrin binding promotes ***cell adhesion.***

(Garver et al, 1997; Tuvia et al 1997)

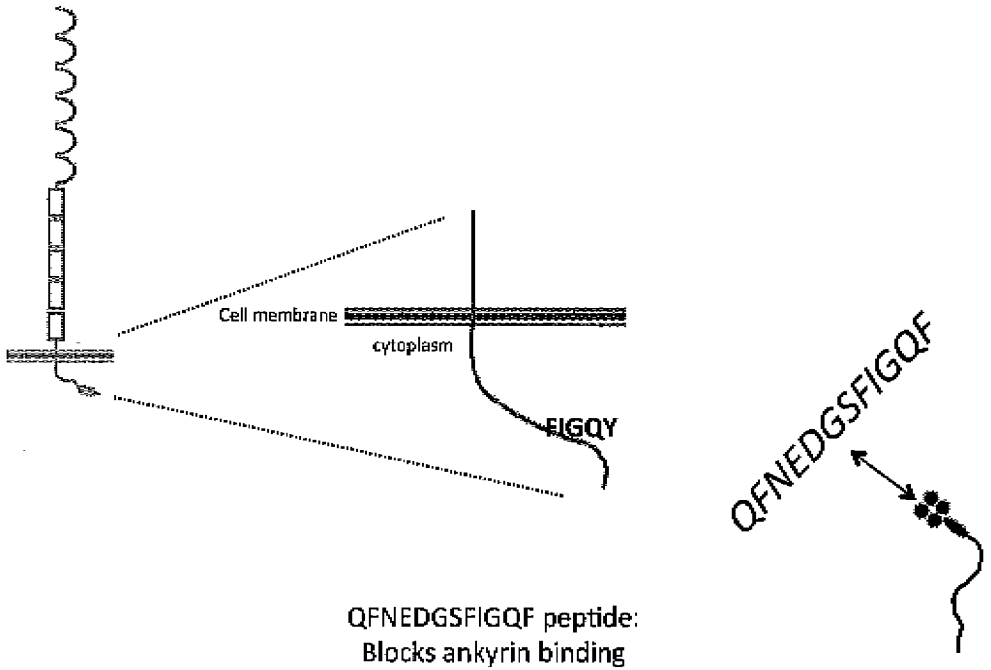
Figure 2. Schematic diagram showing transmembrane & cytoplasmic domain of full-length L1 family members with FIGQF mutation



- FIGQF mutation cannot be phosphorylated
- Binds ankyrin (not regulated)
- Promotes *cell adhesion*
- ***Neurite outgrowth not addressed***

(Garver et al 1997; Tuvia et al 1997)

Figure 3. Schematic diagram showing transmembrane & cytoplasmic domain of full-length wild-type L1 family members with QFNEDGSFIGQF peptide



QFNEDGSFIGQF peptide:
Blocks ankyrin binding
increased neuronal growth

- QFNEDGSFIGQF peptide blocks ankyrin binding to L1-CAM
- ***Neurite outgrowth increases***

(Present application)